RESIDUAL ISCHEMIA AFTER FIRST THROMBOLYSED MYOCARDIAL INFARCTION:THE GISSI2 IRES RESULTS.

The GISSI-2 IRES (Ischemia Residua) Study Group

The incidence of residual ischemia (RI) following thrombolysis was evaluated in 454 unselected pts aged ≤70 years and prospectively enrolled 24 hrs following their first myocardial infarction (MI) in the IRES study. Mean age was 52 ±9 yrs and 87% were male. The MI was anterior in 40%, inferior in 53%, non Q MI in 14%. Randomized treatment was: SK (52%) or tPA (48%) with or without heparin (51% and 49%).
Pts were studied with: continuous clinical and ECG monitoring in the CCU phase (99.7%); 24 hours Holter between the 5th-7th day (94%); off drug symptom-limited exercise test (89%), high dose echo dipyridamole test (80%) and hyperventilation test (89%) before discharge. Signs of RI were found in 8%, 9%, 27%, 24% and 2.8% respectively.
One of the above markers of RI or in-hospital angina (10%) or re-MI (4.6%), occurred in 210/454 (47%) pts. (Cumulative RI). Age, sex, Q or non Q MI, ECG MI location, thrombolytic agent and time to administration, didn't affect the cumulative RI incidence.
The diagnostic policy employed documents, following thrombolysis, a high incidence of RI; the prognostic significance of the latter needs to be assessed.

BENEFICIAL EFFECTS OF SYNCHRONIZED CORONARY VENOUS RETROPERFUSION DURING HIGH-RISK PTCA COMPARED WITH THOSE OF CARDIOPULMONARY BYPASS

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To clarify that synchronized coronary venous retroperfusion (SRP) can maintain systemic circulation through myocardial circulatory support in 8 patients with left main coronary artery disease (>90% stenosis), we evaluated the anginal symptom, ST elevation (STe) in ECG and the changes in systemic hemodynamics during supported PTCA with SRP (Study1) or with cardiopulmonary bypass (CPS, Study2). The effects of supported PTCA on systemic hemodynamics were assessed by the changes in systolic blood pressure (ΔSBP), pulmonary artery enddiastolic pressure (ΔPAEDP), cardiac output (ΔCI) and the tolerance time until the critical hemodynamics.

Results:	No Support	Study1	Study2
Tolerance Time (sec)	38.6±8.8	57.5±7.1*	57.5±7.1*
Occurrence of Angina	4/8	2/8	3/8
ΔSBP (mmHg)	-40.3±16.2	-22.0±13.0*	-18.3±6.5*
ΔPAEDP (mmHg)	11.6±7.4	4.4±4.7*	5.8±3.7*
ΔCI (l/min/m^2)	-0.48±0.41	-0.43±0.50	
Δmax STe (mV)	0.34±0.19	0.15±0.15*	0.29±0.14#

(mean±SD, *P<0.05 vs No Support, #P<0.05 vs SRP)
Both SRP and CPS prevented hemodynamic collapse in all. Although SRP could attenuate myocardial ischemia, CPS did not.
Conclusion: SRP supports not only myocardial circulation but also systemic circulation during high-risk PTCA.

INFLUENCE OF SURGICAL REVASCULARIZATION ON LATE POTENTIALS IN PATIENTS WITH MYOCARDIAL INFARCTION.

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Late potentials (LP) in pts with myocardial infarction (MI) are generally attributed to a dishomogeneous activation of perinecrotic regions, due to the presence of normal myocardium, ischemic myocardium and patchy scar tissue.
We evaluated the influence of myocardial revascularization on the presence of LP in 65 pts (6F, 59M, aged 30-78yrs) with previous MI (ant:16, inf:31, ant+inf:4, non Q:14). Only one pt had ventricular tachycardias. A Marquette MAC 15 HiRes ECG recorder was used for LP identification; 250 beats were averaged and filtered at 40-250 Hz. At least two of the following criteria were required to define LP: duration of high frequency low amplitude signals >38 msec; RMS voltage of terminal 40 msec <20uV , filtered QRS duration > 120 msec. Before revascularization, LP were present in 19 pts (29%); clinical, electrocardiographic, hemodynamic and angiographic findings were similar in both groups. After revascularization, LP disappeared in 7 pts (37%), whereas LP became evident after surgery in 3 pts, one of whom had perioperative MI. No predictive index of disappearance of LP was identified.
These data indicate that the coronary bypass gratting can determine the disappearance of LP, possibly due to functional recovery of ischemic perinecrotic areas; this could in part explain the antiarrhythmic efficacy of myocardial revascularization in pts with VT.

MYOCARDIAL INFARCTION FOLLOWING SUCCESSFUL CORONARY ANGIOPLASTY IS NOT DUE TO INCOMPLETE REVASCULARIZATION.

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It has been suggested that complete revascularization by percutaneous-coronary angioplasty (PTCA) may reduce the risk of subsequent myocardial infarction (MI). However, it is not known if MI occurring late after successful PTCA usually results from an occlusion at the previous PTCA site, occlusion at an undilated stenosis, or occlusion at a previously non-stenotic site. Accordingly, we evaluated 34 patients who suffered 35 MI's more than two weeks (17 to 2055 days) after successful PTCA in which all dilated lesions were reduced to <50% stenosis. The culprit lesion for the subsequent MI was determined by coronary angiography performed within 72 hours of MI. Seventeen (49%) of the MI's occurred in patients who had incomplete revascularization with PTCA leaving one or more >50% stenoses undilated. However, only 3 of the MI's (9%) were due to occlusion at the site of one of these undilated stenoses. Twelve MI's (34%) were due to occlusion at the PTCA site, while 20 MI's (57%) were due to occlusion at a previously non-stenotic, non-PTCA site. In 3 of these patients with culprit lesions that were previously non-stenotic, the MI occurred within 2 months of PTCA.
We conclude that many MI's following successful PTCA are due to occlusion at a site that did not warrant dilation since it did not contain a stenotic lesion at the time of PTCA. Although many of our patients with MI after PTCA had incomplete revascularization, 91% of the MI's could not have been prevented by complete revascularization.